

Asbestos: a chronology of its origins and health effects

R Murray

Abstract

The emotionalised subject of asbestos is treated in chronological terms: how the "magic mineral" known in ancient times in Europe and Asia became in the late nineteenth century an important industrial resource of particular interest to the navies of the world; and how its malign effects gradually became apparent during the present century. The media have made asbestos a notorious villain, but it still has properties and applications useful to society if they are properly controlled in the same way as other industrial hazards. One important application is the manufacture of asbestos cement pipes which are a convenient and cheap method of providing water supplies and sewage disposal for developing countries. An appeal is made for prudence and not hysteria in relation to the use of mineral fibres of all types.

No subject has raised more emotion in the field of occupational health in the past 25 years than asbestos. This is a pity since scientific issues should not become emotional. Unfortunately, the fault lies with ourselves, at least with some of us who ought to know better than to provide the sensation seeking media with ammunition for their misconceived campaigns and the legal profession and the asbestos removal contractors with a rich harvest.

Asbestos: the history of its use

The first difficulty is that the word asbestos is not a scientific but a commercial term applied to a range of mineral fibres that have an application in heat and friction resistant materials such as insulation and brake linings. Originally it did not appear to be a matter of medical concern. Although there were different applications of the various fibres described as asbestos, there was no indication that they might have different pathological manifestations as a result of exposure.

The story is a long one that has been told many

times. Let me just point out the highlights. Deposits of the "magic mineral" in the European Alps have been known since ancient times and the idea of a mineral wool that could be spun and woven commended itself to the Romans, the Vikings, and later the Emperor Charlemagne. Not only was it found in Europe but Marco Polo on his return from his travels described how, in one of the northern provinces of the Great Khan, the inhabitants wove an indestructible cloth from a mineral fibre dug out of the earth.¹ He makes the point that the salamander was not an animal which resisted fire but this mineral fibre. There is no evidence, despite misreadings, deliberate or otherwise, of ancient texts, of any disease being attributed to its use.

Coming to more modern times the story of the Chevalier Jean Aldini (1762-1834) is worth telling as it is not widely known.² He was a professor of physics at the University of Bologna and a nephew of the great Galvani. He led a blameless life, supporting his uncle in his arguments with Volta about the nature of electricity until he retired at the age of 65. He then, apparently suddenly, conceived an interest in fire protection and persuaded a weaver in Lyons to make some asbestos cloth and felt out of which he constructed a suit to protect against fire. This he exhibited in several European capitals, including the Royal Institution in London in 1829, where his discoveries were warmly commended by Michael Faraday. Oddly enough, I have not found any evidence of his ideas being commercialised, though this may have been due, as suggested by Fisher (v infra) to the numerous crises which kept part of Europe in a "perpetual state of disquietude."

The modern exploitation of asbestos began about 100 years ago. In *The Engineer* of 22 June 1883 an article entitled "Asbestos and its applications" has a surprisingly modern look. It refers to the ancient stories and also to the Chevalier Aldini but it is much more concerned with its application to packings for steam engines, which were first introduced by John Bell in 1879 and immediately adopted by the British and German navies. It was also used with soapstone for locomotives, woven into cloth for theatre curtains, and used as a base for filter material. Millboard was manufactured for fire protection and as an electric insulator. Other uses included fireproof cement and putty and the elements for gas fires. The author (unknown) ends by saying: "In conclusion,

we will just say that we have been tempted to dwell rather fully on the nature and applications of this interesting material, as we believe they are not generally known to our readers. . . ."³

Some 10 years later in the *Transactions of the Institute of Marine Engineers* there appeared an extensive article and discussion on "The mining, manufacture and uses of asbestos" by J Alfred Fisher.⁴ He scorns the habit of referring to ancient uses of asbestos and says "I should like to see the valuable space of our technical papers, when devoted to the subject of asbestos, occupied with information brought down to a later date and of a more practical nature. . . ." He refers to the multiple sources of asbestos: Newfoundland, United States, southern and central America, China, Japan, Australia, Spain, Portugal, Hungary, Germany, Russia, the Cape, and Central Africa, but concentrates on what he considers to be the best, Italian and Canadian.

According to Fisher there were two main deposits in Italy, one in the Susa and Aosta valleys in the north west, the other to the north east of Lake Como. In the Susa valley the work was carried on at a height of 6–10 000 feet. The material was brought down the mountainside on a toboggan or sledge and two men could bring down 8 cwt in three hours. In the eastern deposit the mines were at a height of 3600 to 7200 feet where the climate was "comparatively mild." "The inhabitants worked willingly at the asbestos mines, in spite of its not being unattended by danger from landslips and avalanches." The first commercial mine was opened in 1870, the last in 1876. In 1989 the last remaining Italian mine, at Balangero, about 50 km north of Turin, finally closed.

Although Canadian asbestos (which he recognised as chrysotile) had been exhibited at the International Exhibition in London in 1862, no attempt was made to work it for some years. The credit for its discovery goes to a French-Canadian called Fecteau. In the first year of mining operations, 1878, only 50 tons were taken out but by 1884 large and increasing quantities were exported, including, so I have been told, 100 tons in 1880 to Rochdale in England where Samuel Turner, a manufacturer of packings, had his factory. In 1883 several companies were brought together in the United Kingdom as The United Asbestos Company including the earliest which had been established in Glasgow in 1871 as The Patent Asbestos Manufacture Company and which had adopted the salamander as its trademark.

The author refers to more than a hundred uses of asbestos, including the lining of coal bunkers on ships to prevent the effects of spontaneous combustion as a result of heat from the flue gases.

In passing, it is worth noting an article by Wright in which he comments on the danger of cotton silicate (a mixture of cotton fibres and sodium silicate) used for boiler insulation in the British Navy in 1891.⁵

This gave rise to attacks of respiratory irritation. He says "as engineering efficiency improved, steam temperatures were raised and cotton silicate no longer provided sufficient insulation . . . [its] use was discontinued and a substitute employed. The substitute? Asbestos."

History of health effects of asbestos

The first hints of any adverse effect came from the factory inspectors in the United Kingdom and France,^{6,7} but it was in 1899 that a carder from Barking went to see Dr Montague Murray at Charing Cross Hospital. Murray did not report the case, which showed an unusual fibrosis of the lungs, until 1906 when he gave evidence to a departmental committee on compensation for industrial diseases. The patient described how he was the only survivor of 10 men working in the cardroom, all the others having died at ages around 30.

There was no immediate reaction. Indeed the departmental committee was unable to recommend compensation for any occupational pulmonary disease because of the overwhelming complication of tuberculosis.⁸ During the first world war the use of asbestos increased enormously in the navies of the world. Wagner said in 1968 that "in 1916 the two greatest commercial concentrations of crocidolite met at Jutland." He went on to say that "more lives were saved by asbestos that day than have been taken since."⁹

The next time asbestos was mentioned in a medical publication was an article by Cooke in the *British Medical Journal* in 1924.¹⁰ He coined the name asbestosis in 1927 and discovered the "curious bodies" associated with the disease.¹¹ He called them "asbestosis bodies." Subsequent cases in the 1920s were still haunted by the spectre of tubercle but in 1928 Seiler in Glasgow reported a case in which there was fibrosis with no tuberculous complication. This triggered off the interest of E R A Merewether who had been appointed as HM Medical Inspector in 1926. With his engineering inspector colleague C W Price he conducted a survey of the asbestos industry between 1928 and 1930.¹²

This was the first comprehensive study of the health effects of asbestos. It was followed in remarkably quick time by the Asbestos Industry Regulations of 1931 which came fully into force in 1933.¹³ These were the first regulations in any country dealing with the asbestos hazard.

During the 1930s several papers described the disease of asbestosis. Two of the most valuable are those of Merewether in 1933¹⁴ and Gloyne and Merewether in 1938.¹⁵ A few papers described cancer of the lung in asbestos workers. In one Gloyne said "It seemed worth while to record these two cases, not in any attempt to make a case for the aetiological association of these two diseases, but in order to

emphasise certain histological points. . . ."¹⁶ A similar observation was made by Lynch and Smith in the United States.¹⁷ The growing incidence of lung cancer recognised during the war culminated in the study by Bradford Hill and Doll in 1947 implicating cigarette smoking as the primary cause of the disease, though in Germany and Czechoslovakia at the time there were also articles on lung cancer in asbestos workers. Meanwhile as a result of improved housing conditions, diet, and, to some small extent, immunisation with BCG, the incidence of tubercle was gradually dropping and with the introduction of the antibiotics, notably streptomycin, the problem of tubercle gradually diminished to vanishing point. The graphs of mortality from lung cancer and tuberculosis crossed in about 1955.

Perhaps in response to the German and Czech work Merewether had become interested in the possibility of a relation between asbestos and bronchogenic cancer. He asked his medical inspectors, of whom I had become one in 1947, to look at recent postmortem reports on asbestos workers and identify the cause of death. The results were published in his annual report for 1947 which, for various reasons, did not appear until 1949.¹⁸ Because of my privileged position in Manchester—I had three large asbestos factories in my division—my contribution was the greatest numerically, though I am sufficiently modest to admit that I deserve no credit. It was Merewether's idea. I merely did some of the work. It did, however, inculcate an interest in and a curiosity about asbestos that has continued until now.

I discovered that of the last 100 necropsies of asbestos workers, 25 had died of lung cancer. I was sufficiently curious to look at the last 100 necropsies of pottery workers, the "potteries" being also in my area. Only two had died of lung cancer. So much for the recent excitement about silica and lung cancer.

During the 1950s the annual reports of the Chief Inspector of Factories continued to record the number of cases of lung cancer, though these were derived from the work of the pneumoconiosis medical panels rather than from the medical inspectorate. In 1955, following the suspicions generated by Merewether, the role of asbestos in causing lung cancer was established epidemiologically by Doll.¹⁹ He concluded, on the basis of 113 men who had worked for at least 20 years in places where they were exposed to asbestos dust, that lung cancer was a specific hazard of certain asbestos workers and that the average risk among men employed for 20 or more years was 10 times that experienced by the general population. "*The risk,*" he says, "*has become progressively less as the duration of employment under the old dusty conditions has decreased*" (emphasis added). Curiously enough, there is no mention of smoking.

Little attention was paid to this discovery. The number of people in the industry was small and the

main cause of lung cancer was (and still is) the smoking of cigarettes. The outstanding dust problem at the time was coalworkers' pneumoconiosis which was the subject of intensive study by the Pneumoconiosis Research Unit of the Medical Research Council at Cardiff and also by the International Labour Organisation, which produced its first set of radiographs illustrating the International Classification of Radiographs of Pneumoconiosis in 1959. (The most recent revision of the classification was in 1980 to take account of the irregular opacities associated with asbestos. A further revision is likely in 1991 to take account of the pleural changes in asbestos related disease.)

All this was to change, however, after the publication in 1960 of the paper on mesothelioma by Wagner *et al.*²⁰ Here was a rare tumour occurring in relatively large numbers in a circumscribed geographical area where crocidolite was mined and transported. Of the 33 cases, in only eight was asbestos exposure demonstrated. The remainder, excluding one case in which there was no history of exposure to asbestos, had circumstantial evidence of exposure, having lived near the mines or transported the material. There were four "industrial" cases, two who lagged locomotive boilers, one who lagged steam pipes, and one who made fireproof clothing.

The paper took the scientific world by storm and wherever people looked for mesothelioma sure enough they found it, especially in areas where shipyards or asbestos factories had used blue asbestos. The features which made it extraordinary were (1) that the exposure to asbestos was often only environmental and therefore apparently relatively low and (2) the long latent period, up to 40 years in many instances. Cases were found in several countries and these were presented at a symposium at the New York Academy of Sciences in December 1964 under the chairmanship of Irving Selikoff, who had shown asbestos related disease, including mesothelioma, in a group of New Jersey shipyard insulators in 1964. This captured the attention of the world's media and resulted in what I have called a pandemic of mediagenic disease. There was a surprising consensus on the dangers of asbestos and since then it has seldom been out of the news.

The impetus given by the media was apparent in many countries. In the United Kingdom, for example, there was a clamour for legislation that resulted in the Asbestos Regulations of 1969.²¹ The import of crocidolite had been voluntarily abandoned in 1966, the last uses being for making battery boxes for London buses and army tanks. (The introduction of polypropylene had eliminated the need for the acid resisting properties of crocidolite in this particular application.) The British Occupational Hygiene Society in 1968, in response to the growing evidence of the effect of fibres rather

than particles, had produced its standard of 2 fibres/ml as an appropriate level to prevent asbestosis.²²

The flood of papers on asbestos became a torrent. The report of the New York Academy of Sciences in 1965 contained several seminal papers,²³ many of which were analysed by J C Gilson in his Wyers Memorial Lecture of 1965.²⁴

Standard setting for asbestos exposure

At this point, science seemed to go out of the window and a "wage negotiation" approach took its place. Ignoring the fact that biological parameters are geometric rather than arithmetical, an unholy trade in setting standards developed. This was complicated by the cancer factor. Whereas it was possible to argue almost arithmetically on the appropriate standard for silica in causing lung fibrosis, it was not feasible, in view of our ignorance of the nature of the process of malignancy, to extrapolate these figures to a substance causing cancer.

The first extrapolation took place with the guidance notes to the Asbestos Regulations of 1969. The figures of the British Occupational Hygiene Society were related to asbestosis. How could they be related to mesothelioma? Nobody knew. What about a factor of ten, one order of magnitude, it was asked? Why not? So the standard for crocidolite was set at one tenth of the figure for chrysotile and asbestosis—namely, 0.2 f/ml. There it has remained because nobody still knows any better. There were no dust counts, much less fibre counts, in the 1940s and 1950s, so the extraordinary outbreak of mesothelioma has no real numerical foundation.

The argument waxed during the 1970s but it was already clear in 1972 from the work, primarily of Stanton and Wrench,²⁵ that the carcinogenic potency, certainly in experimental animals, depended on the length and diameter of the fibre. Any fibre with a length greater than five microns and a diameter less than three microns could induce cancer if the fibre had sufficient durability and therefore residence time in the lung.

This theory was to be brilliantly and cataclysmically proved by the discoveries of Baris at Karain in Cappadocia between 1975 and 1978.²⁶ Mesothelioma had been endemic in the village since time immemorial but there was no asbestos in the soil. This showed clearly that the question of mesothelioma did not begin and end with crocidolite asbestos and that any fibre, whatever its origin, with appropriate dimensions and durability could cause mesothelioma.

Nothing that has occurred since has altered these findings. Attempts were made to show that the Karain deposits contained amphibole asbestos but, although there is some tremolite in certain areas of Anatolia and in several other places in eastern

Europe, the only fibre in Karain is a volcanic fibre of the zeolite family—erionite or chabazite.

The argument still goes on about what level of asbestos or fibre exposure is acceptable. Extrapolations are bandied about but there has not yet been sufficient time to define the critical dose. That it exists there can be no doubt but we will have to wait until subsequent illness or death can be directly attributed to specific levels of exposure. In view of the long latent period this will take a long time. Meanwhile we must be prudent and not hysterical.

Priorities in relation to asbestos

During the second world war the overwhelming priority was the winning of the war. Anything else was entirely subordinate. Asbestos was seen as a primary protection against fire resulting from enemy attack and so the more asbestos the greater the protection. The United States Maritime Commission underwrote the contracts for the building of Liberty ships, though they have never acknowledged their part in this decision. The result has been a series of claims based on product liability which has fallen on the suppliers of asbestos containing materials. At that time there was no knowledge of lung cancer or mesothelioma and work practices were poor as they were in many industries. It is improper for the apostles of hindsight to suggest that sufficient evidence existed about asbestos as to have been able to anticipate its effects.

Today the priority in developing countries is the supply of potable water and the disposal of sewage. The cheapest and most convenient method in both cases is the provision of asbestos cement pipes. They can be manufactured using local labour and, if not local asbestos, then local sources of cement. There is no point in incurring the expense of polyvinyl chloride or polypropylene or stainless steel pipes in dealing with this problem. When you consider the lives that would be saved by providing clean water and effective sewage disposal and compare this with the minimal risk incurred by miners in exporting countries or factory workers making asbestos cement pipes, I doubt that the finance minister of any developing country would have difficulty in making up his mind.

Conclusion

The asbestos story is really two stories. One is a mythical exaggeration of the media which characterises asbestos as an evil spirit and, ignoring the effect of dose, arrives at the conclusion that "one fibre kills." This popular impression has resulted in politicians, local and national, reacting to the public fear by removing asbestos unnecessarily from schools and public buildings to show that they are doing something. The other story is the true problem of the interface between science (and the refinements of

research which show differences in fibre types and fibre dimensions to be critical) and the intelligent development of public policy and regulation. The essential scientific problem is the inhalation of fibres, whatever they are called or whatever their origin.

Fibre reinforcement, of cement or plastic or bitumen, is a recognised engineering technique that is bound to continue for technical reasons. It is not for us as occupational physicians to preach to industry about what they can and cannot use. It is our clear duty to put them on their guard against the possible dangers and to take the appropriate measures of protection, based on an analysis of the available data and a sense of perspective and proportion.

I submit that, in our current ignorance, we must be careful about the use of any respirable fibre. Having been associated with the asbestos industry since 1947 when I saw my first asbestos necropsy and having seen the changes in the industry during that time I am confident that, although much remains to be discovered, provided that intelligent precautions are taken, the advantages of asbestos or any other fibre reinforcement may be accepted by the people of the world without fear.

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